“ASSESSMENT OF RISK FACTOR FOR DEATH IN DENGUE HAEMORRHAGIC FEVER & DENGUE SHOCK SYNDROME”

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CERTIFICATE

This is to certify that the dissertation titled **"ASSESSMENT OF RISK FACTOR FOR DEATH IN DENGUE HAEMORRHAGIC FEVER & DENGUE SHOCK SYNDROME"** submitted by Dr.*K.Senthil Kumar* to the Faculty of pediatrics, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree(Paediatrics) is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I. **DR.K.Senthil Kumar** solemnly declare that the dissertation titled **"ASSESSMENT OF RISK FACTOR FOR DEATH IN DENGUE HAEMORRHAGIC FEVER & DENGUE SHOCK SYNDROME"** has been prepared by me.

This is submitted to **The Tamilnadu Dr. M.G.R. Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in Paediatrics.

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ABBREVIATIONS

DF	Dengue Fever
DHF	Dengue Haemorrhagic Fever
DSS	Dengue Shock Syndrome
GI	Gastro Intestinal
WHO	World Health Organisation

CONTENTS

INTRODUCTION

Dengue, a Spanish alteration of the Swahili word *Ki-dinga,* is the most common mosquito-born viral illness in humans. The earliest known documentation of dengue like symptoms was recorded in the *Chinese Encyclopaedia of Symptoms* during the Chin Dynasty (AD 265-420). The illness was called "the water poison" and was associated with flying insects near water.

Reason for mortality in DHF/DSS[1] Failure to recognise the patient in shock ,Haemorrhage, Failure to recognise the patient had entered congestive phase may cause cardiac overload and consequent congestive heart failure and death . There has been paucity of data regarding the usefulness of clinical and lab investigation in predicting the death in DHF/DSS.

Hence this study was carried out to study the predictive value of clinical parameters and abnormal laboratory values in dengue illness. The present study may place in perspective the role of clinical parameter and laboratory values in finding out the risk factor leading to death

Dengue Epidemiology

Dengue is the most rapidly spreading mosquito-borne viral disease in the world. In the last 50 years, incidence has increased 30-fold with increasing geographic expansion to new countries and, in the present decade, from urban to rural setting. An estimated 50 million dengue infections occur annually and

approximately 2.5 billion people live in dengue endemic countries[20] . During epidemics of dengue attack rates among susceptible are often 40%-50%, 90% of hospitalised DHF were children less than 15years , mortality in dengue is 5%[21], In India infections are becoming more frequent involvement of younger age group and increased the frequency of epidemics are indicator of higher incidence of infection [2].

The virus[16]

Dengue virus (DEN) is a small single-stranded RNA virus comprising four distinct serotypes (DEN-1 to -4). dengue virus belong to the genus Flavivirus, family Flaviviridae. The mature particle of the dengue virus is spherical with a diameter of 50nm containing multiple copies of the three structural proteins, a host-derived membrane bilayer and a single copy of a positive-sense, single-stranded RNA genome. The genome is cleaved by host and viral proteases in three structural proteins (capsid, C, prM, the precursor of membrane, M, protein and envelope, E) and seven nonstructural proteins (NS).

The vectors[16]

The various serotypes of the dengue virus are transmitted to humans through the bites of infected *Aedes* mosquitoes, principally *Ae. aegypti*. This mosquito is a tropical and subtropical species widely distributed around the world, mostly between latitudes 35 0N and 35 0S. The immature stages are found in water-

filled habitats, mostly in artificial containers closely associated with human dwellings and often indoors. Studies suggest that most female *Ae. aegypti* may spend their lifetime in or around the houses where they emerge as adults. This means that people, rather than mosquitoes, rapidly move the virus within and between communities. The eggs can remain viable for many months in the absence of water

The host[16]

After an incubation period of 4--10 days, infection by any of the four virus serotypes can produce a wide spectrum of illness, although most infections are asymptomatic or subclinical . Primary infection is thought to induce lifelong protective immunity to the infecting serotype . Individuals suffering an infection are protected from clinical illness with a different serotype within 2--3 months of the primary infection but with no long-term cross-protective immunity. Individual risk factors determine the severity of disease and include secondary infection, age, ethnicity and possibly chronic diseases (bronchial asthma, sickle cell anaemia and diabetes mellitus). Young children in particular may be less able than adults to compensate for capillary leakage and are consequently at greater risk of dengue shock.

Transmission of the dengue virus[16]

Humans are the main amplifying host of the virus. Dengue virus circulating in the blood of viraemic humans is ingested by female mosquitoes during feeding. The virus then infects the mosquito mid-gut and subsequently spreads systemically over a period of 8--12 days. After this extrinsic incubation period, the virus can be transmitted to other humans during subsequent probing or feeding. The extrinsic incubation period is influenced in part by environmental conditions, especially ambient temperature. Thereafter the mosquito remains infective for the rest of its life. *Ae. aegypti* is one of the most efficient vector for arboviruses because it is highly anthropophilic, frequently bites several times before completing oogenesis, and thrives in close proximity to humans. Vertical transmission (transovarial transmission) of dengue virus has been demonstrated in the laboratory but rarely in the field.

Pathogenesis[11]

The pathogenesis is incompletely understood, but epidemiologic studies suggest that it is usually associated with secondary infections with dengue types 1–4. Early in the acute stage of secondary dengue infections, there is rapid activation of the complement system. Shortly before or during shock, blood levels of soluble tumor necrosis factor receptor, interferon-γ, and interleukin 2 are elevated. C1q, C3, C4, C5–C8, and C3 proactivators are depressed, and C3

catabolic rates are elevated. These factors may interact at the endothelial cell to produce increased vascular permeability through the nitric oxide final pathway. The blood clotting and fibrinolytic systems are activated, and levels of factor XII (Hageman factor) are depressed. The mechanism of bleeding in dengue hemorrhagic fever is not known, but a mild degree of disseminated intravascular coagulation, liver damage, and thrombocytopenia may operate synergistically[11]. Capillary damage allows fluid, electrolytes, small proteins, and, in some instances, red cells to leak into extra vascular spaces. Plasma leakage is thought to be associated with functional rather than destructive effects on endothelial cells[16]. Activation of infected monocytes and T cells, the complement system and the production of mediators, monokines, cytokines and solublereceptors may also be involved in endothelial cell dysfunction This internal redistribution of fluid, together with deficits caused by fasting, thirst, and vomiting, results in hemoconcentration, hypovolemia, increased cardiac work, tissue hypoxia, metabolic acidosis, and hyponatremia. Microscopically, there is perivascular edema in the soft tissues and widespread diapedesis of red cells. There may be maturational arrest of megakaryocytes in bone marrow, and increased numbers of them are seen in capillaries of the lungs, in renal glomeruli, and in sinusoids of the liver and spleen.

Clinical Features:

In endemic areas DHF is more commonly seen in children below 15 years. The major clinical manifestations seen in typical cases of DHF are high fever, hemorrhagic phenomena, hepatomegaly and often circulatory failure. Thrombocytopenia and hemoconcentation are distinctive laboratory findings. WHO has given guidelines for case definition and grading of DHF.

Case definition of DHF:

This is based on two clinical criteria (fever and bleeding) and two laboratory criteria (low platelet count and vascular leak).

1. Acute onset, high grade, continuous fever for 2-7 days.
2. One of the following hemorrhagic manifestations

   A positive tourniquet test

   Petechiae, Purpura or ecchymosis

   Epistaxis, gum bleeding, GI bleed (hematemesis and / or melena)
3. Evidence of plasma leak:

   A rise in hematocrit (Hct) equal to or greater than 20%

   Pleural effusion, ascites, or hypoalbuminemia or hypoproteinemia
4. Thrombocytopenia (Platelet count < 1,00,000/ mm^3)

Grades of DHF:

DHF is divided into 4 grades. The first two grades are characterized by the criteria mentioned above. Grade III and IV are characterised by shock.

Grade I DHF: Criteria for DHF mentioned above with hemorrhagic manifestation as positive tourniquet test only

Grade II DHF: Criteria for DHF + skin / mucosal bleeding

Grade III DHF: DHF Criteria with circulatory failure characterized by rapid and weak pulse, narrow pulse pressure (20 mm Hg or less) or hypotension with cold clammy skin and restlessness.

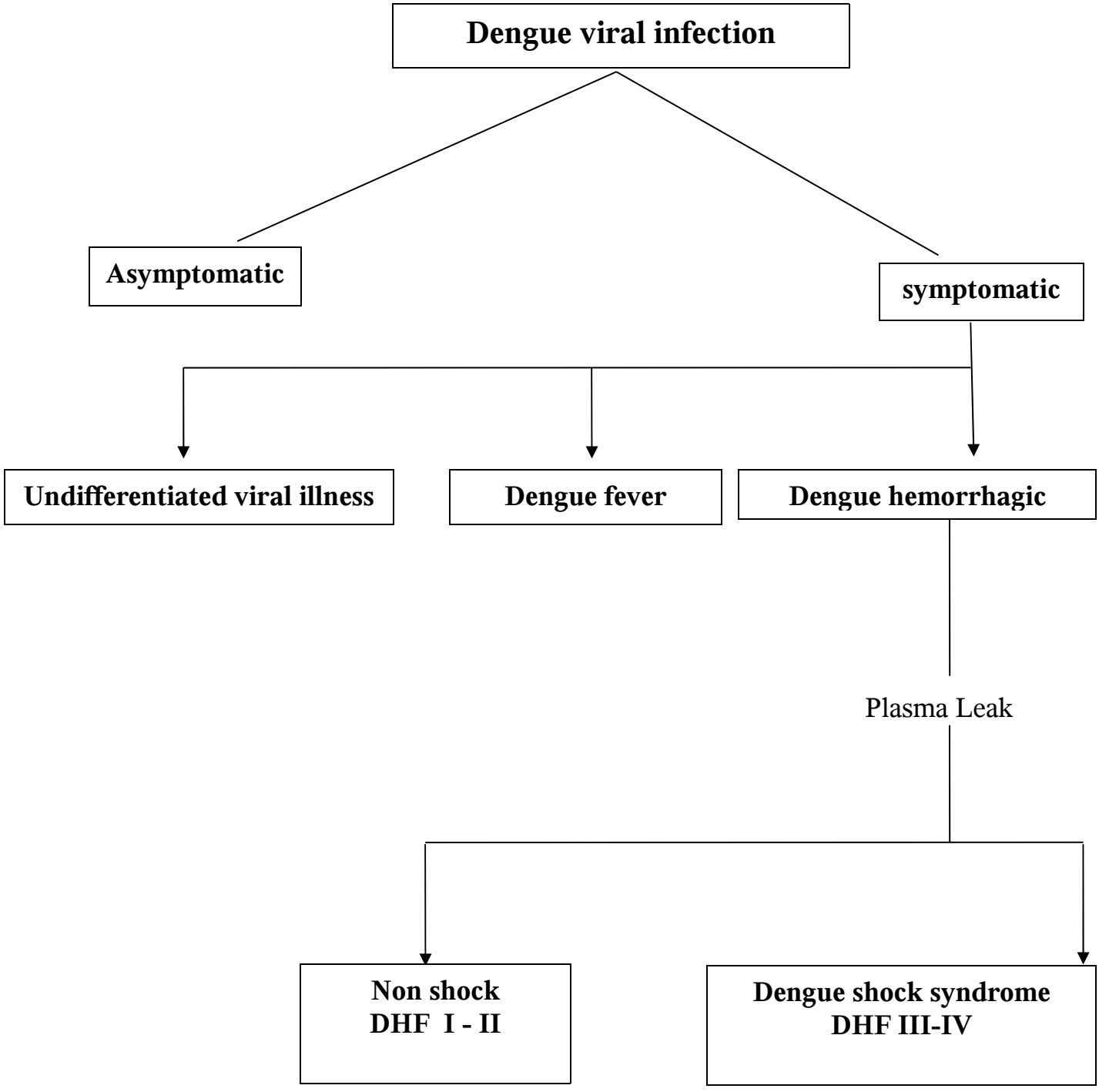
Grade IV DHF: DHF with profound shock with undetectable pulse and blood pressure.

Clinical illness in DHF is characterized by 3 phases (1) Acute febrile phase (2) Critical phase and (3) Convalescent Phase. [24]

Febrile phase

Patients typically develop high-grade fever suddenly. This acute febrile phase usually lasts 2–7 days and is often accompanied by facial flushing, skin erythema, generalized bodyache, myalgia, arthralgia and headache . Some patients may have sore throat, injected pharynx and conjunctival injection. Anorexia, nausea and vomiting are common.

Figure 1. Manifestation of dengue viral infection



monitoring for warning signs and other clinical parameters is crucial to recognizing progression to the critical phase.

Mild haemorrhagic manifestations like petechiae and mucosal membrane bleeding (e.g. nose and gums) may be seen The liver is often enlarged and tender after a few days of fever . The earliest abnormality in the full blood count is a progressive decrease in total white cell count, which should alert the physician to a high probability of dengue.

Critical phase

Around the time of defervescence, when the temperature drops to 37.5–38°C or less and remains below this level, usually on days 3–7 of illness, an increase in capillary permeability in parallel with increasing haematocrit levels may occur . This marks the beginning of the critical phase. The period of clinically significant plasma leakage usually lasts 24–48 hours.Progressive leukopenia followed by a rapid decrease in platelet count usually precedes plasma leakage. At this point patients without an increase in capillary permeability will improve, while those with increased capillary permeability may become worse as a result of lost plasma volume. Pleural effusion and ascites may be clinically detectable depending on the degree of plasma leakage and the volume of fluid therapy. Hence chest x-ray and abdominal ultrasound can be useful tools for diagnosis. The degree of increase above the baseline haematocrit often reflects the severity of plasma leakage.

Shock occurs when a critical volume of plasma is lost through leakage. It is often preceded by warning signs. The body temperature may be subnormal when shock occurs. With prolonged shock, the consequent organ hypoperfusion results in progressive organ impairment, metabolic acidosis and disseminated intravascular coagulation. This in turn leads to severe haemorrhage causing the haematocrit to decrease in severe shock. Instead of the leukopenia usually seen during this phase of dengue, the total white cell count may increase in patients with severe bleeding. In addition, severe organ impairment such as severe hepatitis, encephalitis or myocarditis and/or severe bleeding may also develop without obvious plasma leakage or shock

Recovery phase

If the patient survives the 24–48 hour critical phase, a gradual reabsorption of extravascular compartment fluid takes place in the following 48–72 hours. General well-being improves, appetite returns, gastrointestinal symptoms abate, haemodynamic status stabilizes and diuresis ensues. Some patients may have a rash of “isles of white in the sea of red” . Some may experience generalized pruritus. Bradycardia and electrocardiographic changes are common during this stage.

The haematocrit stabilizes or may be lower due to the dilutional effect of reabsorbed fluid. White blood cell count usually starts to rise soon after defervescence but the recovery of platelet count is typically later than that of

white blood cell count, Respiratory distress from massive pleural effusion and ascites will occur at any time if excessive intravenous fluids have been administered. During the critical and/or recovery phases, excessive fluid therapy is associated with pulmonary oedema or congestive heart failure.

Diagnostic Tests in Dengue Infection

There are 3 methods to be looked at

(1) Serology (2) Isolation of virus (3) Demonstration of specific virul antigen or viral RNA in serum.

Serology[25]

Most widely used serologic test is IgM capture by ELISA technique (MAC – ELISA). It is simple and rapid test. By 6-10 days 93% have detectable IgM antibodies, MAC ELISA test requires only a single properly timed blood sample after 5 days of fever. Still a negative test does not rule out dengue

Two types of serologic responses are seen : Primary and secondary. For acute and convalescent serum capture IgM (MAC – ELISA) and IgG ELISA are now standard for detection and differentiation of primary and secondary DV infection. [25] In primary dengue infection, IgM levels an higher. In secondary dengue infection IgG levels are higher and IgM is low or absent. Recently by capture IgE Elisa, it is shown that DV specific IgE titres are significantly higher in DHF and / or DSS patients than in patients with DF. [25]

Viral Isolation

Circulating DV levels early in illness correlate with increasing dengue severity. Viral isolation can be done by mosquito inoculation or culture. But it is generally not available in many places .

Molecular Diagnosis

Real time one step RNA PCR is the new gold standard for rapid diagnosis of dengue infection. [25] It is a simple, rapid method with low contamination rate. But the cost is high and is generally not available.

Antigen Detection

DV has 3 structural protein genes and 7 non-structural protein (NS) genes. Non Structural protein NSI antigen assay by dot blot immue assay in serum and plasma samples of suspected DV infected patients is widely evaluated for the diagnosis and prediction of severity of dengue infection.

Principles in Management of DHF and DSS[21,24]

1. DSS is a hypovolemic shock due to plasma loss accompanied by an increase in peripheral vascular resistance. The period of short, but life threatening. The Severity of shock is variable.
2. The critical period of plasma leak with varying degrees of circulatory disturbances occurs during the 48 hours around defervescence of fever (about 24 hours before and 24 hours after the fall of temperature). The critical period may be seen anytime from third day after onset of fever.

The vascular leak is limited to a period of about 48 hours after which the leaked fluid will be reabsorbed into vascular space. If fluids are given inappropriately or for longer duration than necessary, serious volume overload and pulmonary oedema can occur. Balancing fluid administration to prevent shock with avoidance of excessive administration reqires intensive skilled monitoring.

3. During the critical period, the child can go in and out of shock many times necessitating close monitoring and adjustment of fluid administration.

In a child with fever following warning signs are explained to parents to bring the child immediately to the hospital

Child appears ill, irritable or lethargic as fever subsides

Vomiting and abdominal pain

Very thirsty or refusal to eat / drink

Sweating

Cold extremities

Decreased urine output

Skin bleeds in the form of petechiae or purpura or any bleeding.

Indications for admission

Child is very weak , cannot eat / drink

Any bleeding other than petechiae

Platelet count less than 10,0000 /and or increase in Hct 10-20% or pleural effusion or ascites.

Other clinical features of shock / impending shock

Infants

Underlying disease condition. E.g. Heart disease

Parental concern / difficult to follow up

Management of DSS

Only about 1/3 of patients with DHF develop shock. They should be managed in an intensive or semi intensive care unit which should also be mosquito free.

Judicious volume replacement is necessary in DSS. Shock is a medical emergency necessitating prompt and immediate replacement with fluids. Good supportive care, frequent monitoring and adjustment of fluid administration are the keys to successful outcome. Airway and respiration are maintained and oxygen is administered at highest concentration possible, usually through a non-rebreathing mask.

Types of IV fluids and blood products used

Normal saline (NS) or Ringer's lactate (RL) for initial resuscitation of shock; 5% dextrose in RL, NS or ½ NS after correction of shock.

Hyperoncotic colloids such as dextran – 40/70 or 6% Hydroxy ethyl starch (hetastarch) or gelatin in refractory shock.

Fresh whole blood or packed RBC's – for significant bleed.

FFP – in DIC

Platelets – only in certain specific situations.

For initial resuscitation of shock which may require repeated boluses isotonic crystalloids (NS or RL) are preferred. Only after shock correction, dextrose containing solutions are used. Frequent monitoring of vital signs and urine output with Hct if available, will guide in step up or step down of IV fluids.

Monitoring

Pulse, BP, capillary refill time (CRT), consciousness and respiration should be monitored every 30-60 minutes or more frequently if necessary during the first 1 to 2 hours and then less frequently after the child is stabilized.

Urine output has to be chartered hourly with an indwelling urinary catheter.

Micro Hct, if available, should be checked initially 1-2 hourly and later less frequently as child improves. This is a good indicator of plasma leak and reliable guide for further fluids.

Other lab tests such as blood grouping and cross matching; blood sugar, blood gases, serum electrolytes and calcium, renal function tests and coagulogram (PT, PTT) are done as per clinical situation.

Fluid Therapy

In grade I – II DHF 5% dextrose in NS or RL in the form of maintenance fluid plus fluid deficit of 5-8% is needed initially. This works out to 6ml/kg/hr. This is gradually stepped down and stopped in 12-24 hours.

In Grade III DHF, NS or RL can be administered at 10-20 ml/kg/ over 1 hour (WHO recommends 5% GNS or 5% dextrose in RL 10ml/kg over 1 hour). This bolus an be repeated. If clinical signs of shock improve rate of fluid administration is stepped down gradually. If shock worsens, treatment is given as for grade IV DHF.

In grade IV DHF (pulse and BP not recordable), 20ml /kg of NS or RL is infused rapidly till pulses appear and repeated if necessary. As the child improves, rate of fluid administration is stepped down with periodic monitoring. This protocol is based on resuscitation of shock as per PALS guidelines and also many recent studies that have tried various fluid options.

If shock persists colloids such as dextran-40 or hetastarch or gelatin at a rate of 10ml/kg/hour is administered. Hct estimation is helpful in further management. If Hct is still high and shock persists, repeat colloids at 10ml/kg/hour upto 30ml/kg. If Hct is falling and the child is in shock, concealed hemorrhage is suspected and fresh whole blood 10ml/kg or

Figure 2.Volume replacement flow chart for patients with DHF grade 1 and 2

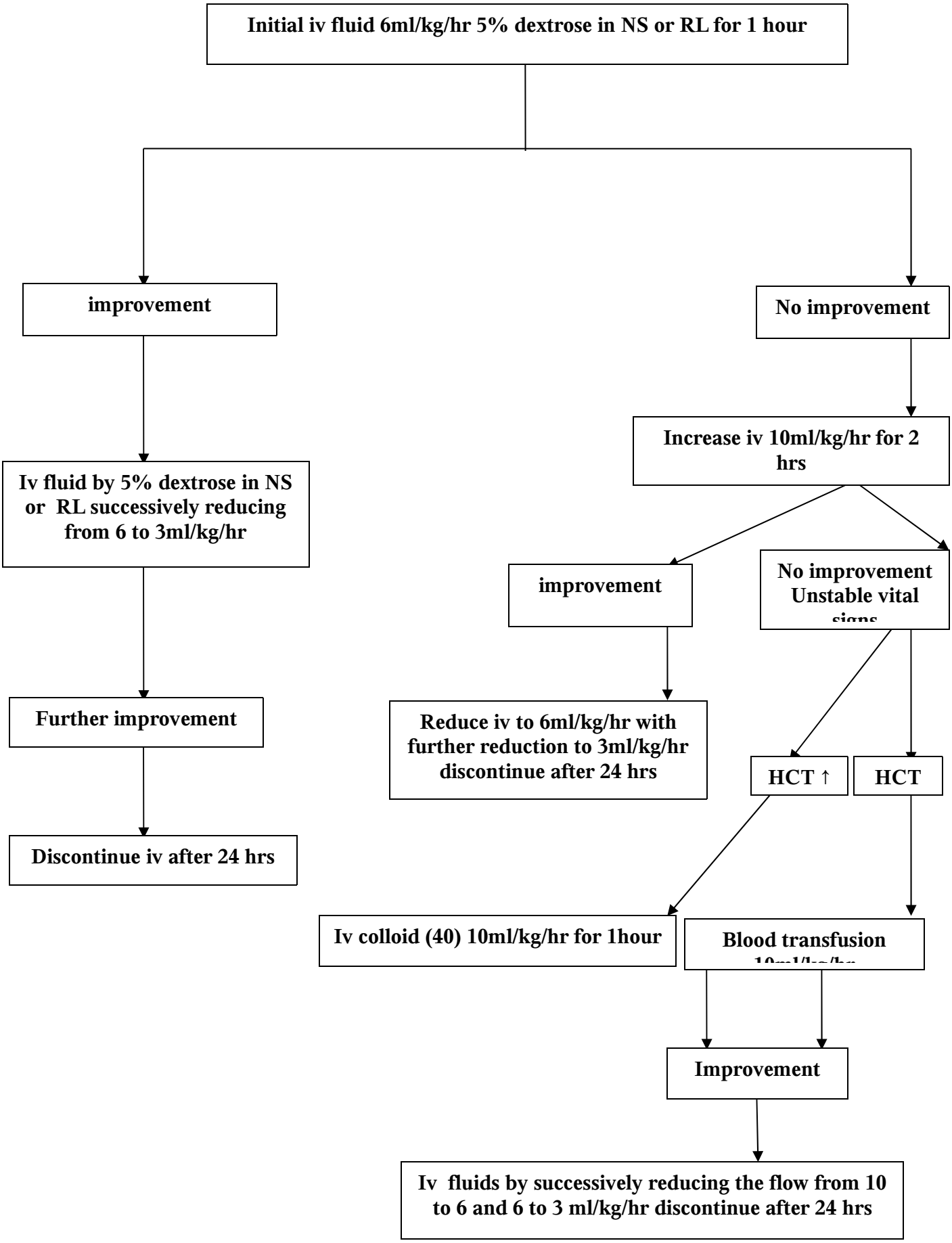
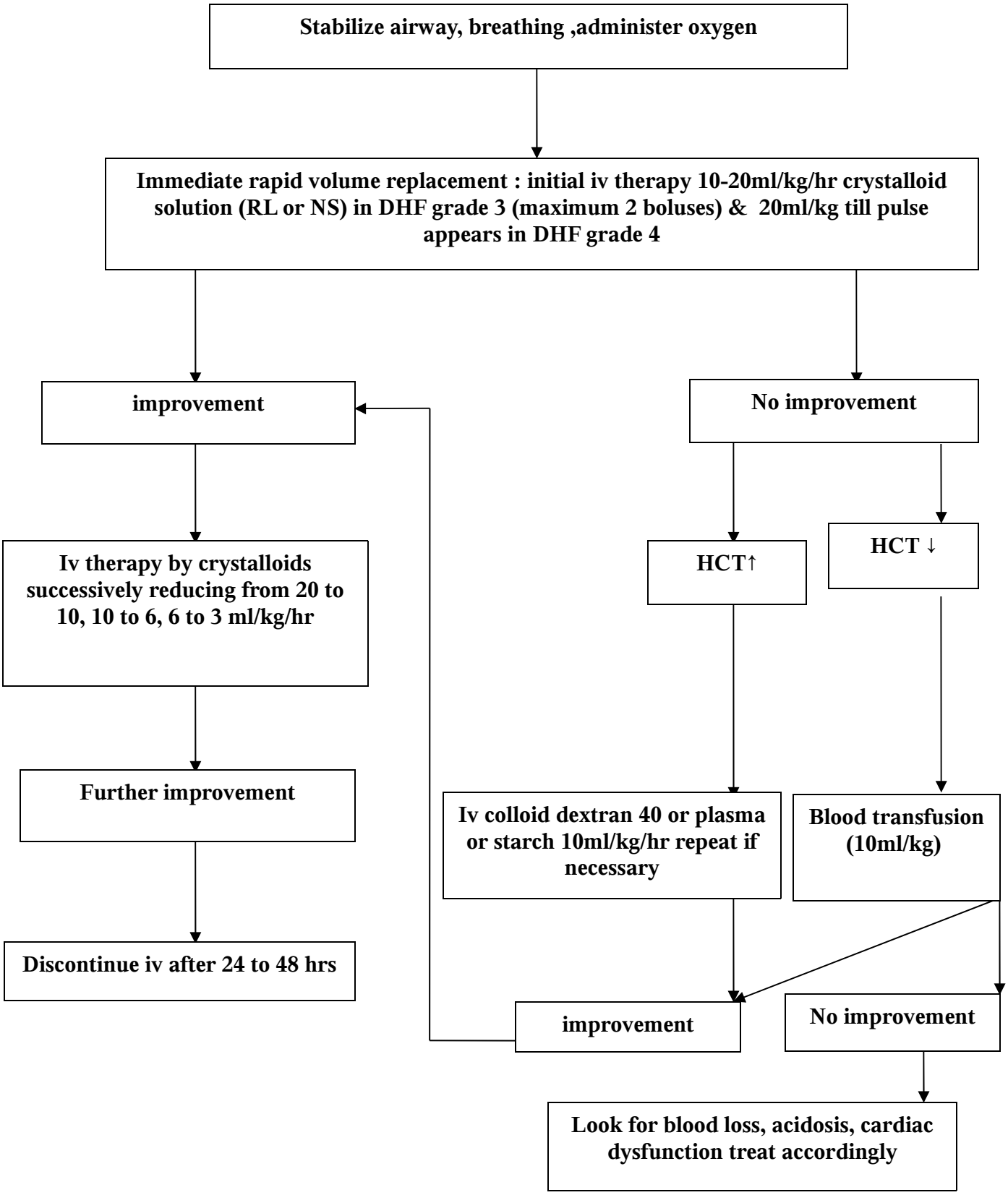


Figure 3.Volume replacement flow chart for patients with DHF 3 & 4



packed RBC's 5ml/kg is infused (Fig.3). Transfusions can be repeated as necessary. Volume and type of fluids are to be charted on ½ - 1 hourly basis till shock is corrected.

crystalloid infusion is adequate for early resuscitation. In DHF IV (severe shock) colloid may be preferred.

If child improves clinically and has good urine output IV fluid rate is gradually decreased and stopped over a period of 24 hours. IV fluid administration should be discontinued when Hct decreases to stable level of 40% and child's appetite returns and urine out put is sufficient. In general fluid therapy is not required after 48 hours of treatment of shock. It is very important to recognize the improvement in child's status and appropriately reduce / stop IV fluids as there is also restoration of extraverted fluid in convalescent stage.

Most patient of DHF/DSS without severe bleed have good prognosis. A serious pitfall in severe shock is failure to recognize concealed internal hemorrhage and continued transfusion of colloid instead of blood / packed RBC's. This may lead on to volume overload and respiratory failure with high mortality. Major clinical challenge in DHF is management of DHF/DSS with prolonged shock often complicated by massive bleeding.

Children who do not improve with above measures and are in refractory shock are likely to have severe metabolic problems, pulmonary oedema and DIC. They need escalated therapy in the form of invasive monitoring like

central venous pressure (CVP), inotropes and ventilatory support Based on CVP, fluids, blood components and inotropes are infused. A recent study had used bedside ECHO cardiogram to assess volume status and cardiac function (systolic and diastolic dysfunction) for administration of fluids, inotropes and vaso-active agents in refractory cases and careful fluid removal by controlled diuresis or dialysis in the event of extensive oedema with better results.

Indication for colloids

Colloids are indicated in patients who continue to be in shock with high Hct despite receiving adequate volume of crystalloid and patients who have signs of fluid overload eg. Respiratory distress from massive pleural effusion and / or very tense abdomen It is preferable to use colloids which are hyperoncotic such as dextran-40, hydroxyethyl starch or gelatin (Hemaccel) than plasma as they are more efficient, cost effective and blood related complications do not occur. Maximum dosage of colloids used is 30 ml/kg. Colloids many interfere with hemostais and higher doses may cause transient elevation of urea, creatinine or cause acute renal failure.

Indications for blood transfusion

- In patients with severe bleeding such as GI bleeding.
- In patients who have persistent shock after receiving adequate crystalloid with decreasing Hct when internal bleeding is suspected.
- Concealed internal bleeding with prolonged shock.

Fresh whole blood 10ml/kg or packed RBCs 5ml/kg is administrated. Additional transfusions may be required based on Hct and clinical signs.

Indications for platelets In DHF, bleeding may be due to vascular involvement, thrombocytopenia, platelet functional abnormality and / or coagulaopathy. Platelets are administered..

- If there is major bleeding.
- When platelet counts is less than 50000/cmm and if any invasive procedure is planned
- In absence of bleeding when platelet count is less than 10000/cmm.

I unit platelet concentrate / 10 kg body weight will raise platelet count by about 10000 platelets should be infused rapidly in about 10 minutes. This may lead onto volume overload when many units are given.

When DIC is present FFP, platelet concentrate and cryoprecipitate may be required.

In DSS about 60% require crystalloid, 20% need colloid and about 15% blood components. Only 0.4% require platelets.

Complications of DSS

1. Electrolyte disturbances and metabolic problems (hyponatremia, hypocalcemia, hypoglycemia, acidosis) are to be identified and corrected.
2. DIC may occur after prolonged shock and uncorrected academia and

may cause massive bleed and lethal shock. Monitor PT, PTT and fibrin dehydration productions (FDPs) to identify DIC during shock.

3. Fluid overload can occur both during critical and convalescent phases. This is serious complication as it may lead onto acute pulmonary oedema, congestive heart failure, later respiratory failure and death. Administration of correct type of IV fluids in just adequate amounts to maintain effective circulation based on frequent monitoring particularly hourly urine output will prevent this complication.

Treatment of fluid overload will involve close monitoring of vitals and urine output, ventilatory support if needed, CVP monitoring, administration of colloid such as dextran-40 and careful administration of IV frusemide, preferably by infusion upto 0.4 mg/kg/hr.

4. Noscocomial infection such as gram negative sepsis, pneumonia and urinary tract infection. Children who have profound shock (unrecordable BP), massive GI bleed and shock with delayed admission may have mortality more than 50%.

Certain unusual manifestations of DHF are increasingly reported in recent years

1. Encephalopathy: This occurs more commonly in infants. It may occur due to prolonged shock, intracranial bleed, acute hepatic encephalopathy, electrolyte imbalance, vascular occlusion and rarely due to dengue virus

causing encephalitis. Seizures, altered sensoirum, spasticity and paresis may result.

2. Acute hepatic failure
3. Myocarditis: Global hypokinesia and decreased ejection fraction may result. Arrhythmias, heart blocks, and bradycardia may occur.

Mortality in DHF/DSS is due to

1. Prolonged shock
2. Fluid overload
3. Massive bleeding which is secondary to prolonged shock and acidosis leading on to DIVC
4. Acute hepatic failure and encephalopathy

Mortality rates reported are from <1% to 5% for DHF in centres experienced in resuscitation. Mortality reports from Indian literature suggest case fatality rate of 26-47%.

DIFFERENTIAL DIAGNOSIS [11]

The differential diagnosis of dengue fever includes viral respiratory and influenza-like diseases, the early stages of malaria, mild yellow fever, scrub typhus, viral hepatitis, and leptospirosis. Four arboviral diseases have dengue-like courses but without rash: Colorado tick fever, sandfly fever, Rift Valley fever, and Ross River fever

Prevention[11]

Several types of dengue type 1–4 vaccines are under development, . Prophylaxis consists of avoiding mosquito bites by use of insecticides, repellents, body covering with clothing, screening of houses, and destruction of *A. aegypti* breeding sites. If water storage is mandatory, a tight-fitting lid or a thin layer of oil may prevent egg laying or hatching. A larvicide, such as Abate [O,O′-(thiodi-p-phenylene) O,O,O,O′-tetramethyl phosphorothioate], available as a 1% sand-granule formation and effective at a concentration of 1 ppm, may be added safely to drinking water. Ultra-low-volume spray equipment effectively dispenses the adulticide malathion from truck or airplane for rapid intervention during an epidemic. Only personal antimosquito measures are effective against mosquitoes in the field, forest, or jungle.

The possibility exists that dengue vaccination may sensitize a recipient so that ensuing dengue infection could result in hemorrhagic fever. Vaccination with yellow fever 17D strain has no effect on the severity of dengue illness, although seroconversion rates to a dengue type 2 vaccine were enhanced in persons immune to yellow fever.

Review Of Literature

1. Vinod H rategeri et al. [5] department of paediatrics, Karnataka institute of medical sciences, hubli . done a study, in 23 children on clinical profile and outcome of dengue treatment . 35% were infants , severity more noticed in females , common clinical features noted in order of frequency were fever (100%), vomiting (82%) , abdominal pain (67%), restlessness 65% , headache 22%.
2. Kalyanarooj et al[6] queen sirikit national institute of child health Bangkok. studied on 4532 cases observed that malnourished children had a higher risk of developing shock and high case fatality rate .
3. Batra prerna et al[10] department of paediatrics, mahatma Gandhi institute of medical sciences sevagram maharastra . studied 499 patients and found that children who were referred late, had profound shock and unconsciousness , were expired within 24hours of admission.
4. Banik et al[12] department of paediatrics and virology medical college and school of tropical medicine Calcutta, conducted study on 72 children. causes of death included bleeding 47%, shock 35%, both 18%. In more severe disease abdominal pain , hepatomegaly and ascitis were usually associated .
5. Eric .C.M van grop et al [7]. Conducted a study on 50 children, found that

total cholesterol were decreased in patients with severe disease.

6. Sivabalan et al chennai[8]. Conducted a study on 60 children and fond that raised ALT, tender hepatomegaly abdominal pain , abdominal distension and respiratory distress were significant predictors of bleeding.
7. Anuradha et al[14] . conducted a study in dengue, found that bleeding manifestation in DSS was associated with higher mortality.
8. Chua mn et al[9] conducted a study in dengue and found that patients with a platelet count of <50,000 had a six fold increase in mortality.
9. Bridget et al from oxford university clinical research unit [13] conducted study on 383 children and found ringer lactate to be more useful in moderately severe shock, colloid (starch) to be little more advantage in severe shock. Use of dextran was questioned because of its side effects.
10. Ranjit,el al[17] .conducted study on DSS , Patients with dengue shock syndrome are at high risk of mortality due to refractory shock and multiple organ failure. Aggressive shock management and possibly the use of judicious fluid removal may decrease mortality rates in the severest forms of dengue shock syndrome.
11. N. M. Dung et al[18] conducted study on Fluid Replacement in Dengue Shock Syndrome: A Randomized, Double-Blind Comparison of Four Intravenous-Fluid Regimens**.** Dextran 70 provided the most rapid normalization of the hematocrit and restoration of the cardiac index,

without adverse effects, and may be the preferred solution for acute resuscitation in DSS.

12. Shrishu R. Et al[19] . Conducted study on Clinical features, complications and atypical manifestations of children with severe forms of dengue hemorrhagic fever in South India it was found that complications such as DIC, diastolic dysfunction, abdominal compartment syndrome, ARDS and hepatic dysfunction were more frequent in severe established shock. Children referred late were harder to resuscitate. There were 9 PICU deaths (case fatality rate of 8.35%). Severe refractory shock, DIC, ARDS, hepatic failure and neurological manifestations singly or in combination were the commonest causes of death.
13. K. Yusoff, et al [23] National University of Malaysia and Lumpur, Malaysia Abnormal ECG seen in 15 patients (65%) consisted of. Abnormal echocardiograms were present in 12 patients (52%); these were pericardial effusion, abnormal systolic and diastolic functions, left ventricular dilatation, and tricuspid regurgitation ECG and echocardiographic abnormalities are common during the acute phase of dengue haemorrhagic fever. Cardiac involvement in the pathogenesis of the more severe forms of dengue remains to be defined .

Study justification

Untreated cases of DHF & DSS carry a mortality of 20% (3), but early recognition and management dramatically reduces the mortality to less than 1 % Dengue, an arboviral infection , which occur in epidemics, should be continuously evaluated for any change in current situation . There is no study available from our institute for the assessment of risk factors for death in DHF & DSS

Aim of the study

To assess the risk factor for death in DHF & DSS among children admitted in tertiary referral hospital

Subjects and methods

Study design : nested case control

Case - children who were diagnosed as DHF & DSS and expired

Control - children who were diagnosed as DHF & DSS and discharged well

Study place : institute of child health and hospital for children Egmore Chennai

Period of study: October 2007 to September 2009

Study population: children less than 12 years

Inclusion criteria:

All Children who satisfy WHO criteria for DHF & DSS[11]

Fever

Minor or major hemorrhagic manifestation

Thrombocytopenia (platelet < 1,00,000/mm^3)

Objective evidence of increased capillary permeability

(HCT >20% of initial level, pleural effusion in chest x ray, hypoalbuminemia)

Exclusion criteria:

Co infection of dengue with leptospirosis, typhoid, malaria, etc

Children treated outside hospital with IV fluids or inotropes

Children who went against medical advice .

Sample size:

Total number of DHF & DSS cases during study period

Percentage of least expected prevalent risk factor in case and control = 1:3

Manoeuvre

After getting informed consent, clinical data are collected and entered in the proforma (annexure 1) which includes details about the case history, clinical findings, lab investigations . these patients are divided in to two groups ie patients who were expired and patients who were discharged well

The clinical and lab data of DHF & DSS patients are analysed and compared between these two groups. Various clinical and lab parameters are analysed to assess their value as risk factors for death.

The following parameters are compared between two groups

Age: since the reported mortality rate in previous studies is higher in < 1 year, so infants are compared with older children for mortality risk

Sex: severity of disease is more noticed in female , so it is analysed to assess whether gender is a risk factor for death

Nutrition Status: severe under nutrition is associated with higher risk for death and hence analysed for risk of death

Time from onset of fever to presentation to hospital:

more than 5 days is compared with less than 5 days for risk of death

Clinical status at presentation

Shock in DHF & DSS is associated with high mortality , so presence and absence of shock is assessed for risk factor for death, in shock fluid refractory is associated with high mortality so fluid refractory shock with fluid responsive shock is assessed for risk factor for death

Hematocrit

Hematocrit > 37.5 is associated with risk of bleeding and death , so its risk factor for death is assessed with hematocrit < 37.5

Platelets

Platelets< 50000 is associated with 6times increase in mortality, so platelets <50000 is compared with platelets >50000 for risk factor for death

ALT

More than 3 times normal is associate with high risk for bleeding and death , so its risk for death is analysed with ALT less than 3 times normal

Total cholesterol

Value less than 90.8 mg is associated is risk of death. So its risk for death is analysed with cholesterol value more than 90.8

Colloids

Colloids not given to the sever DSS has high mortality , so it is assessed for risk factor for death

Blood products (FFP/ prbc)

It is analysed to assess weather not giving blood product in appropriate time is associated with mortality

Inotropes

It is analysed to assess weather not giving inotropes in appropriate time is associated with mortality

Risk Factors Analysing in DHF/DSS for death

1. Age
2. Sex
3. Nutritional status
4. Time of presentation to hospital
5. Clinical state at presentation - shock
6. Fluid refractory shock
7. Hematocrit > 37.5
8. Platelet <50,000
9. ALT > 3 times normal
10. Total cholesterol < 90.8 mg
11. Colloid
12. Blood products
13. ionotropes

Statistical analysis

Data was entered in Microsoft office excel and analyzed using SSPS ver.11.0 for windows

Descriptive statistics like frequencies and percentage were obtained for categorical variables association between categorical variables were determined using chi-square test

Univariate ordinal regression with death as the outcome variable was done . this provided an odds ratio for each of the risk factor for death. In order to determine how the significant risk factors are acting independently a multiple ordinal regression model was developed . variables that were significant in chi-square test and those that had a theoretical importance were included in the model

For all the above statistical procedures , significance was determined at 5%

0BSERVATIONS

Total cases studied : 125

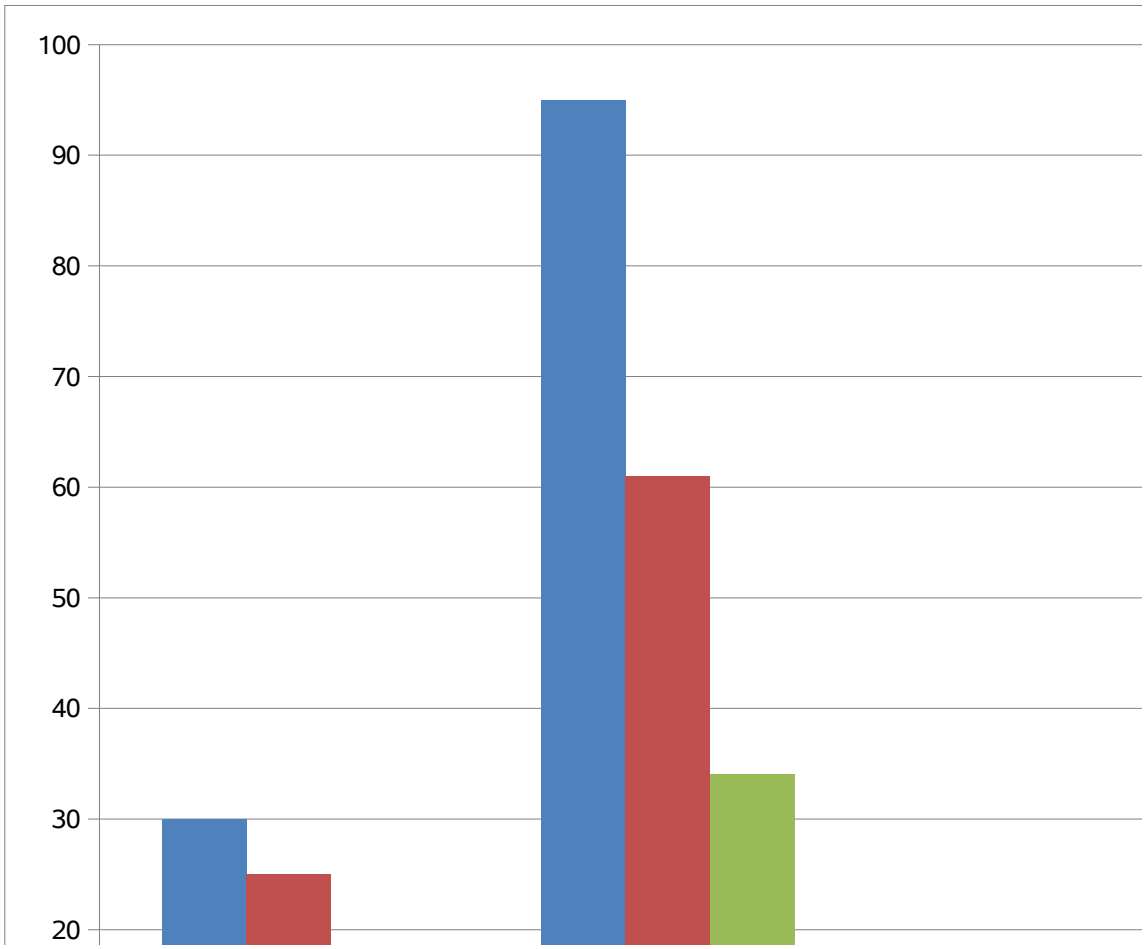
No of cases expired : 30

No of case recovered : 95

Table- 1

In this study mortality in DSS is 42%, DHF is 7% overall mortality out of 125 cases studied is 24% , in this study death death occur on an average time of 48 hrs after hospitalisation (with maximum of 10 days to minimum of 6 hrs after hospitalisation)

Total number of cases in DHF & DSS



Age less than 1 year & more than 1 year in DHF & DSS

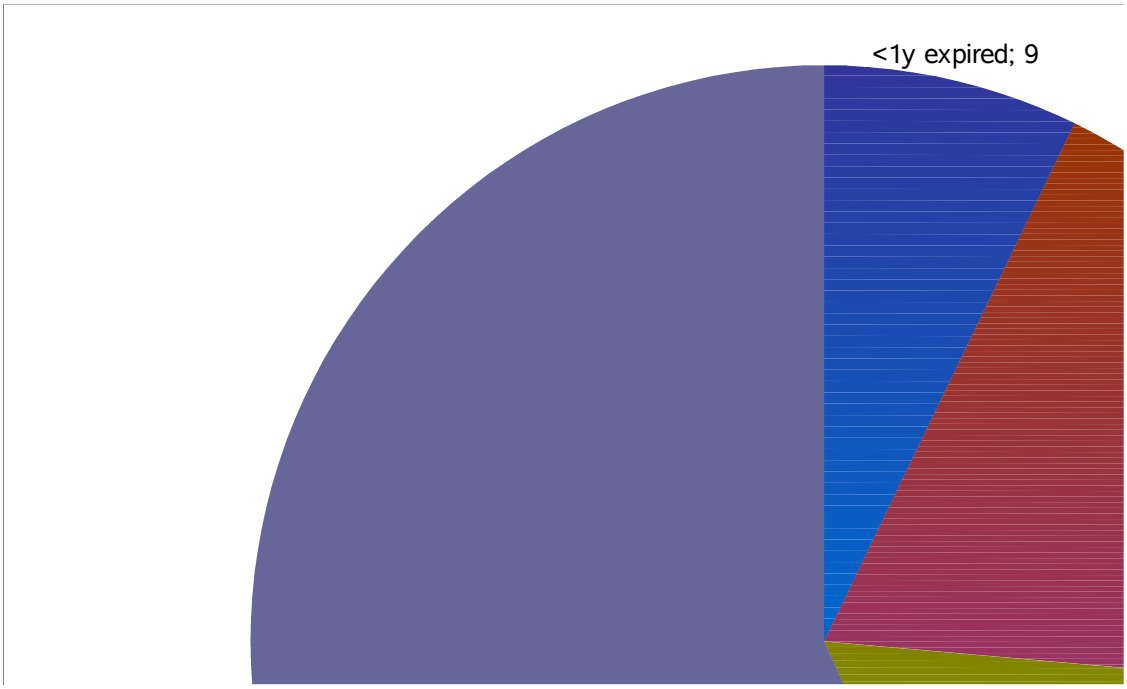


Table-2 .Age and sex

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Age	<1 yr	9(30%)	24(25.3%)	33(26.4%)	1.268	0.15,3.1	0.608
	>1 yr	21(70%)	71(74.7%)	97(73.6%)			
Sex	Female	19(63.3%)	45(47.7%)	64(51.2%)	1.917	0.82,4.4	0.127
	Male	11(36.7%)	50(52.6%)	61(48.8%)			

In this study below 1 year constitute 26.4 % and 73.6% constitute above 1 year. Odds of children expired less than 1 year is 1.26 when compared to those recovered which is not statistically significant OR(95% CI) =

1.26(0.6,2.8). Odds of children expired having female gender is 1.9 , when compared to those who recovered which is not statistically significant OR(95% CI) = 1.9(0.82,4.4)

Table 3 Nutritional status and Fever in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Nutritional status	G3/ G4	4(13.3%)	20(82.1%)	24(19.2%)	0.57	0.18,1.8	0.349
	N/G1/G2	26(86.7%)	75(17.9%)	101(80.8%)			
Fever	>5 days	19(63.3%)	31(32.6%)	50(40%)	3.6	1.5,8.4	0.003
	<5 days	11(36.7%)	64(67.4%)	75(60%)			

Odds of children expired in severely malnourished is 0.57 when compared to those recovered which is not statistically significant OR(95% CI) = 0.57(0.8,1.8) Odds of children expired with fever more than 5 days is 3.6 when compared to those recovered which is statistically significant OR(95% CI) = 3.56(1.5, 8.4)

Table 4 Symptom analysis in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Abdomen Pain	**present**	8(26.7%)	23(24.2%)	31(24.8%)	1.13	0.4,2.9	0.786
	absent	22(73.3%)	72(75.8%)	94(75.2%)			
Abdomen distension	**present**	6 (20%)	14(14.7%)	20(16%)	1.44	0.5,4.1	0.493
	absent	24(80%)	81(85.3%)	105(84%)			
Vomiting	**present**	11(36.7%)	44(46.3%)	55(44%)	0.67	0.2,1.5	0.353
	absent	19(63.3%)	51(53.7%)	70(56%)			
Diarrhoea	**present**	5(16.7%)	12(12.6%)	17(13.6%)	1.38	0.4,4.3	0.574
	absent	25(83.3%)	83(87.4%)	108(86.4%)			

Symptom analysis in DSS & DHF

Skin bleeds	**present**	8(26.7%)	12(12.6%)	20(16%)	2.51	0.9,6.9	0.068
	absent	22(73.3%)	83(87.4%)	105(84%)			
Mucosal bleeds	**present**	2(6.7%)	3(3.2%)	5(4%)	2.19	0.3,13.7	0.393
	absent	28(93.7%)	92(96.8%)	120(96%)			
G.I bleeds	**present**	12(40%)	24(25.3%)	36(28.8%)	1.97	0.8,4.6	0.120
	absent	18(60%)	71(74.4%)	89(71.2%)			

Symptom analysis in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N =125 n (%)	OR	95% C.I	P Value
Convulsions	**present**	16(53.3%)	25(26.3%)	41(32.8%)	3.2	1.3,7.4	0.006
	absent	14(46.7%)	70(73.7%)	84(6.2%)			
Breathlessnes	**present**	15(50%)	17(17.9%)	32(25.6%)	4.5	1.8,11.1	0.000
	absent	15(50%)	78(82.1%)	93(74.4%)			
Oedema	**present**	10(33.3%)	19(20%)	29(23.2%)	2.0	0.8,4.9	0.131
	absent	20(66.7%)	76(80%)	96(76.8%)			
Lethargy	**present**	24(80%)	52(54.7%)	76(60.8%)	3.3	1.2,8.8	0.013
	absent	6(20%)	43(45.3%)	49(39.2%)			

- In patients expired most common symptom other than fever is lethargy 80%, second most common symptom is convulsion 53.3%, least common symptom is mucosal bleed 6.7%
- In patients recovered most common symptom other than fever is lethargy 54.4%, second most common symptom is vomiting 46.3%, least common symptom is mucosal bleed 3.2%

- Odds of children expired having abdomen pain is 1.1 when compared to those recovered which is not statistically significant OR(95% CI) = 1.1(0.4, 2.9)
- Odds of children expired having abdomen distension is 1.4 when compared to those recovered which is not statistically significant OR(95% CI) = 1.4(0.5, 4.1)
- Odds of children expired having vomiting is 0.67 when compared to those recovered which is not statistically significant OR (95% CI) = 0.67(0.2, 1.5)
- Odds of children expired having diarrhoea is 1.3 when compared to those recovered which is not statistically significant OR (95% CI) = 1.3(0.4, 4.2)
- Odds of children expired having skin bleed is 2.5 when compared to those recovered which is not statistically significant OR (95% CI) = 2.5(0.9, 6.9)
- Odds of children expired having mucosal bleed is 2.1 when compared to those recovered which is not statistically significant OR (95% CI) = 2.1(0.3, 13.7)
- Odds of children expired having gastrointestinal bleed is 1.9 when compared to those recovered which is not statistically significant OR (95% CI) = 1.9(0.8, 4.6)

- Odds of children expired having convulsions is 3.2 when compared to those recovered which is statistically significant OR (95% CI) = 3.2(1.3, 7.4)
- Odds of children expired having breathlessness is 3.2 when compared to those recovered which is statistically significant OR (95% CI) = 3.2(1.8, 11.1)
- Odds of children expired having oedema is 2 when compared to those recovered which is not statistically significant OR (95% CI) = 2(0.8, 4.9)
- Odds of children expired having lethargy is 3.3 when compared to those recovered which is statistically significant OR (95% CI) = 3.3(1.2, 8.0)

Table 5 Shock in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Peripheral temperature	Cold	26(86.7%)	41(43.2%)	67(53.6%)	8.5	2.7,26.4	0.000
	Warm	4(13.3%)	54(56.8%)	58(46.4%)			
Peripheral Pulse	Not felt	19(63.3%)	18(18.9%)	37(29.6%)	7.3	2.9,18.2	0.000
	Felt	11(36.7%)	77(81.1%)	88(70.4%)			
shock	Present	27(90%)	47(49.5%)	74(59.2%)	9.1	2.6,32.3	0.000
	absent	3(10%)	48(50.5%)	51(40.8%)			

variables		Expired N = 27 n (%)	Recovered N = 47 n (%)	Total N = 74 n (%)	OR	95% C.I	P Value
If Shock present	Fluid refractory	25	31	56	6.45	3.05,13.3	0.02
	Fluid responsive	2	16	18			

- Odds of children expired having cold periphery is 8.5 when compared to those recovered which is statistically significant OR (95% CI) = 8.5(2.7, 26.4)
- Odds of children expired having absent peripheral pulse is 7.3 when compared to those recovered which is statistically significant OR (95% CI) = 7.3(2.9, 18.2)
- Odds of children expired having shock is 9.1 when compared to those recovered which is statistically significant OR (95% CI) = 9.1(26, 32.3)
- Odds of children expired having fluid refractory shock is 6.4 when compared to those recovered which is statistically significant OR (95% CI) = 6.4(3.0, 13.3)

Table 6 Clinical sign in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Hepatomegaly	Present	29(96.7%)	90(94.7%)	119(95.2%)	1.6	0.18,14.3	0.666
	Absent	1(3.3%)	5(5.3%)	6(4.8%)			
splenomegaly	Present	6(20%)	15(15.8%)	21(16.8%)	1.3	0.46,3.8	0.591
	Absent	24(80%)	80(84.2%)	104(83.2%)			
unconsciousness	Present	24(80%)	47(49.5%)	71(56.8%)	4.0	1.5,10.8	0.03
	Absent	6(20%)	48(50.5%)	54(43.2%)			

- Odds of children expired having hepatomegaly is 1.6 when compared to those recovered which is not statistically significant OR (95% CI) = 1.6(0.18, 14.3)
- Odds of children expired having spleenomegaly is 1.3 when compared to those recovered which is not statistically significant OR (95% CI) = 1.3(0.4, 3.8)
- Odds of children expired having unconscious is 4.8 when compared to those recovered which is statistically significant OR (95% CI) = 4.8(1.5, 10.8)

Table 7 Hematocrit and platelets in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Hematocrit	> 37.5	11(36.7%)	51(53.7%)	62(49.6%)	0.4	0.21,1.1	0.104
	< 37.5	19(63.3%)	44(46.3%)	63(50.4%)			
Platelets	< 50,000	26(86.7%)	78(82.1%)	104(83.2%)	1.4	0.43,4.5	0.560
	> 50,000	4(13.3%)	17(17.9%)	21(16.8%)			

- Odds of children expired having hematocrit more than 37.5 is 0.4 when compared to those recovered which is not statistically significant OR(95% CI) = 0.49(0.21, 1.1)
- Odds of children expired having platelets less than 50,000 is 1.4 when compared to those recovered which is not statistically significant OR(95% CI) = 1.4(0.4, 4.5)

Table 8 ALT ,cholesterol in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
ALT	> 3times	23(76.7%)	52(54.7%)	75(60%)	2.7	1.0,6.9	0.03
	< 3 times	7(23.3%)	43(45.3%)	50(40%)			
cholesterol	< 90.8	6(20%)	0(0%)	6(4.8%)			0.00
	> 90.8	24(80%)	95(100%)	119(95.2%)			

- Odds of children expired having ALT more than 3 times is 2.7 when compared to those recovered which is statistically significant OR (95% CI) = 2.7(1.0, 6.9)

Table 9 Radiology in DSS & DHF

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Chest X ray	cardiomegaly	14(46.7 %)	3(3.2%)	17(13.6%)	26.8	6.9,104	0.000
	No cardiomegaly	16(53.3 %)	92(96.8%)	108(86.4 %)			
USG	Ascitis	18(60%)	73(76.8%)	91(72.8%)	0.15	0.18,1.08	0.071
	No ascitis	12(40%)	22(23.2%)	34(27.2%)			

- Odds of children expired having cardiomegaly is 26.8 when compared to those recovered which is statistically significant OR(95% CI) = 26.8(6,9, 104)
- Odds of children expired having ascitis is 0.15 when compared to those recovered which is not statistically significant OR(95% CI) = 0.15(0.18, 1.08)

Table 10 Treatment in DHF & DSS

variables		Expired N = 30 n (%)	Recovered N = 95 n (%)	Total N = 125 n (%)	OR	95% C.I	P Value
Colloids	given	14(46.7%)	13(13.7%)	27(21.6%)	2.6	2.6,11.2	0.03
	Not Given	16(53.3%)	82(86.3%)	98(78.4%)			
Ffp / prbc	given	12(40%)	18(18.9%)	30(24%)	2.85	1.4,5.6	0.02
	Not Given	18(60%)	77(81.1%)	95(76%)			
Initial ionotropes	given	24(80%)	31(32.6%)	55(44%)	8.2	4.6,16.2	0.01
	Not given	6(20%)	64(67.4%)	70(56%)			

- Odds of children expired received colloids is 5.5 when compared to those recovered which is statistically significant OR (95% CI) = 5.5(2.6, 11.2)
- Odds of children expired received FFP/PRBC is 2.85 when compared to those recovered which is statistically significant OR (95% CI) = 2.85(1.4,5.6)
- Odds of children expired received ionotropes is 8.2 when compared to those recovered which is statistically significant OR(95% CI) = 8.2(4.6, 16.2)

Table 11 Risk factor for death in DHF & DSS derived by univariate logistic regression analysis

variable		OR	95% CI	p- value
fever	> 5 days	3.5	1.5, 8.4	0.003
	< 5 days			
convulsion	Present	3.2	1.3, 7.4	0.006
	Absent			
breathlessness	Present	4.5	1.8,11.1	0.000
	Absent			
lethargy	Present	3.3	1.2, 8.8	0.013
	Absent			
Shock	Present	9.1	2.6, 32.3	0.000
	Absent			
F.refractory shock	Present	6.4	3.0, 13.3	0.02
	Absent			
Peripheral temp	Cold	8.5	2.7, 26.4	0.000
	Warm			
Peripheral pulse	Absent	7.3	2.9, 18.2	0.000
	Present			
unconcious	Present	4.0	1.5, 10.8	0.03
	Absent			
ALT	> 3 times	2.7	1.0, 6.7	0.03
	< 3 times			
cholesterol	<90.8			0.00
	>90.8			
CXR cardiomegaly	Present	26.8	6.9,104	0.000
	Absent			
colloid	Given	5.5	2.6,11.2	0.03
	Not given			
FFP/ prbc	Given	2.85	1.4,5.6	0.02
	Not given			
Inotropes	Given	8.2	4.6,16.2	0.01
	Not given			

Table 12 Risk factor for death in DHF/DSS derived from multiple logistic regression analysis

variable		OR	95% CI	P value
fever	> 5 days	7.9	1.2, 52.4	0.031
	< 5 days			
Convulsion	present	21.8	2.3, 199.9	0.006
	absent			
Breathlessness	present	11.1	1.73, 71.2	0.0111
	absent			
CXR cardiomegaly	present	75	5.6, 1006.04	0.0011
	absent			

Among the various factors which were analysed previously , fever > 5days OR(95%CI) = 7.9(1.2,52.4), Convulsion OR(95%CI) = 21.8(2.3,199.9), breathlessness OR(95%CI) = 11.1(1.7,71.2), CXR cardiomegaly OR(95%CI) = 75(5.6,1006.4) are found to be the independent risk factor for death in DHF & DSS

Discussion

- In our study, infants less than 1 year is26.4%,were as in the previous study 35% were infants , Age and gender have no value in predicting death in dengue hemorrhagic fever and dengue shock syndrome which is comparable to the study done previously[5]
- In our study, mortality in dengue hemorrhagic fever and dengue shock syndrome is more in females (63.3%), so severity of disease is more in females in dengue hemorrhagic fever and dengue shock syndrome , which is comparable to the study done previously Vinod H rategeri et al. [5]
- In our study, lethargy (80%), convulsion (53.3%) in dengue hemorrhagic fever and dengue shock syndrome are associated with high mortality which is comparable to study done previously patients who were referred late had profound shock and unconsciousness , expired within 24hours of admission Batra prerno et al [10].
- In present study, children presented late to the hospital with fever (63.3%)in dengue hemorrhagic fever and dengue shock syndrome were associated with increase mortality which is comparable to study done previously referred late expired within 24hours of admission Batra prerno et al [10].
- In our study, among expired children in dengue hemorrhagic fever and dengue shock syndrome 90% were presented with shock which is

comparable to the study done previously who had profound shock , expired within 24hours of admission Banik et al Calcutta[12].

- In present study, fluid refractory shock in dengue hemorrhagic fever and dengue shock syndrome were associated with high mortality which is comparable to the others study done previously Patients with dengue shock syndrome are at high risk of mortality due to refractory shock and multiple organ failure Ranjit,el al[17].
- In our study, cold periphery and absent peripheral pulses were present in 30% of cases in dengue hemorrhagic fever and dengue shock syndrome associated with high mortality which is comparable to the study done previously Banik et al Calcutta[12].
- In our study, fever is present in 100% of patients with dengue which is comparable to the study done previously Ayyub m et al [15]
- In our study ,mortality in dengue hemorrhagic fever and dengue shock syndrome in severely malnourished children is less than well nourished children which is not comparable to the study done previously malnourished children had a higher risk of developing shock and high case fatality rate kalyana rooj et al [6]
- In our study, platelet has no role in predicting the mortality in dengue haemorrhage fever and dengue shock syndrome which is not comparable to the other study done previously Chua mn et al[9]

- In our study, Total cholesterol less than 90.8 mg is associated with high mortality in dengue hemorrhagic fever and dengue shock syndrome which is comparable to previous study Eric .C.M van grop et al [7].
- In present study, Hematocrit >37.5 was not associated with risk for mortality in dengue hemorrhagic fever and dengue shock syndrome, which is not compared to the previous study in that hemoconcentration is associated with increase risk of bleeding and subsequent mortality Sivabalan et al chennai[8].
- In our study, alanine transaminases level more than 3 times normal is associated with increased risk for mortality in dengue hemorrhagic fever and dengue shock syndrome Which is comparable to the previously done studied elevated transaminases level increase risk of bleeding and subsequent mortality Sivabalan et al chennai[8].
- In our study, requirement of colloids , blood products, and inotropes is a risk factor for mortality, which is comparable to the previous study Aggressive shock management may decrease mortality rate Ranjit,el al[17].
- In our study, cardiomegaly in chest x ray is 43.3% in expired children is associated with increased risk of mortality which is comparable with the previous study ECG and echocardiographic abnormalities are common during the acute phase of dengue haemorrhagic fever. Cardiac involvement in the pathogenesis of the more severe forms of dengue k. Yusoff et al [23]

Summary and conclusion

In our study the following factors are found to be significantly associated with death in DHF/DSS

1. Presented to hospital with fever > 5 days
2. Convulsion
3. Breathlessness
4. Cardiomegaly in Chest x ray

Proper history ,good clinical examination and periodic monitoring the patients would help us to identify patients at risk for bleeding and there by institute pompt treatment and reduce the mortality associate with DHF/DSS

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ANNEXURE 1

Data Entry Form

Name	address
Age	IP number
Sex	date of admission
Weight	date of onset of illness
Height	similar illness in family or locality

Clinical features

Clinical feature	Yes	No	Duration
fever			
Headache			
Myalgia			
Arthalgia			
Retro orbital pain			
Abdomen pain			
convulsion			
Vomiting			
Diarrhea			
Skin bleeds (petechiae)			
Mucosal bleeds(gum bleeds, epistaxis)			
GI bleed(hematamysis, malena)			
Other symptoms			

Signs

Signs	Yes	No
Pallor		
Icterus		
Edema		
Heart rate		
Respiratory rate		
CRT >2 sec		
Periphery cold		
Peripheral pulse absent		
Unconsciousness		
Lymphadenopathy		
Air entry equal in lungs		
Added sounds in lung		
S3 gallop in CVS		
Abdomen distension		
Hepatomegaly		
Spleenomegaly		
CNS alert		
Verbal		
Pain responsive		
unresponsive		

Lab investigation

Haemoglobin	
Total count – wbc	
Differential count	
Platelet	
Hematocrit	
Electrolytes	
Sugar	
Urea	
Creatinine	
AST	
ALT	
Proteins	
Total cholesterol	

Chest xray	
Ultra sonogram	
Serology	

Treatment given

Treatment	yes	No
Crystalloids		
Colloids		
Blood		
Platelets		
Inotropes		
others		

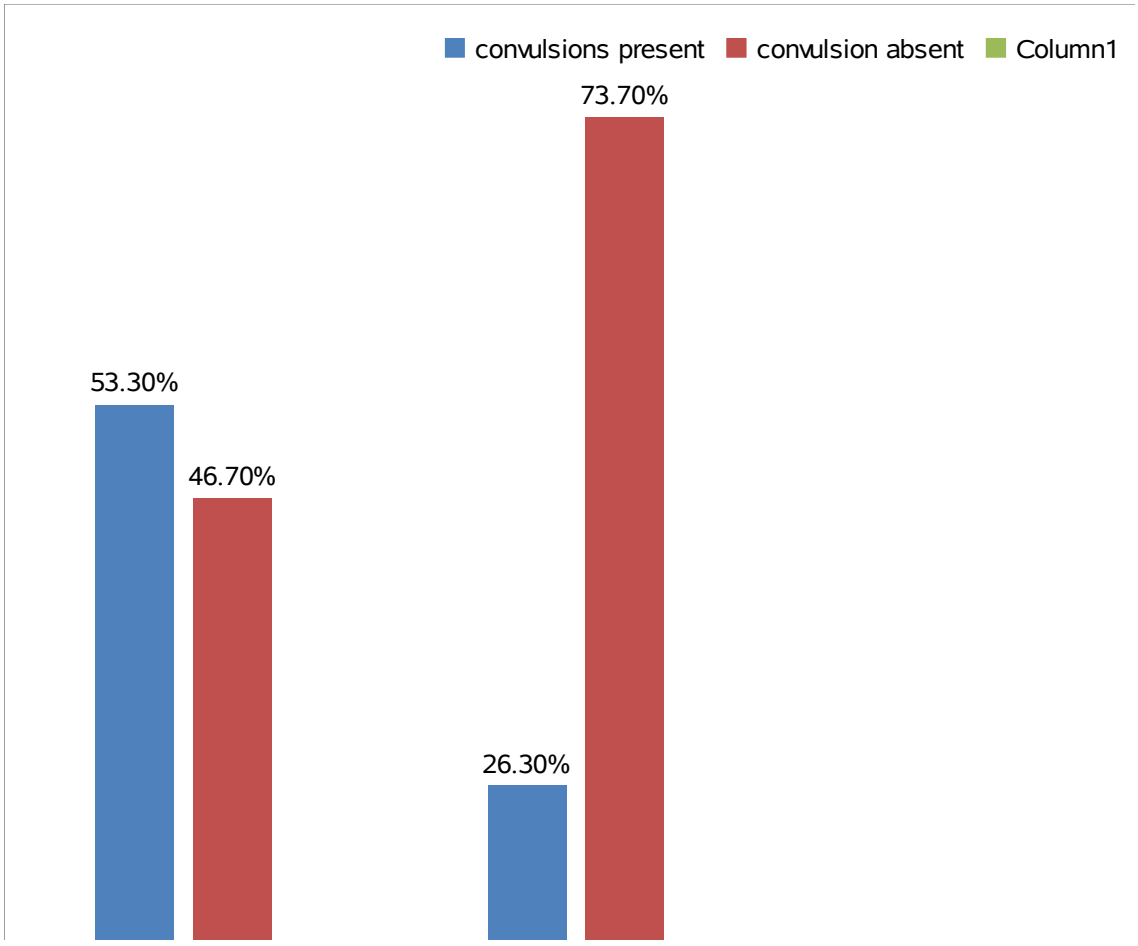
Outcome

Recovered

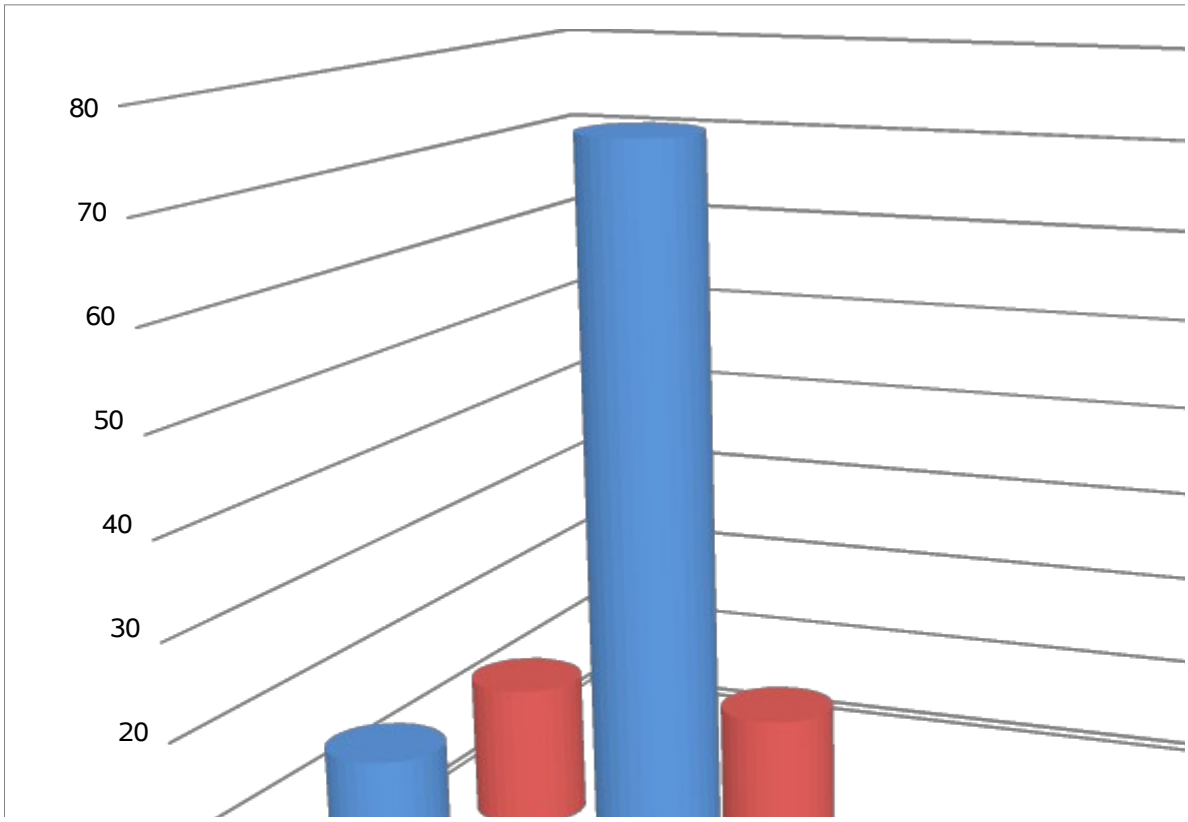
Death

Fever in DHF & DSS in expired group

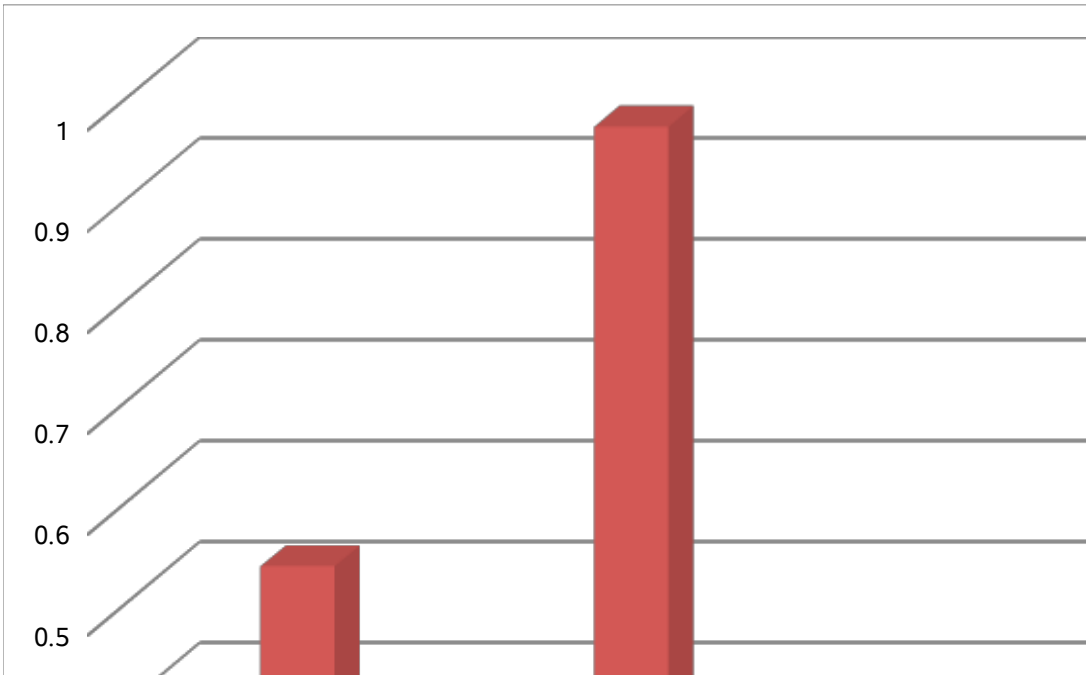
convulsions in dengue hemorrhagic fever and dengue shock syndrome



Breathlessness in dengue hemorrhagic fever and dengue shock syndrome



Cardiomegaly in DHF & DSS


1
0.9
0.8
0.7
0.6
0.5